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Problems in the Estimation of Human Exposure to Components of Acid Precipitation Precursors

by Benjamin G. Ferris, Jr.,* and John D. Spengler*

Problems associated with estimation of human exposure to ambient air pollutants are discussed. Ideally, we would prefer to have some indication of actual dose. For most pollutants this is not presently feasible. Specific problems discussed are adequacy of outdoor monitors; the need to correct for exposures and time spent indoors; the need to have particle size distributions described and the chemistry of the particles presented. These indicate the need to develop lightweight accurate and reliable personal monitors.

Environmental concerns for acid deposition have focused on ecological endpoints. Too little attention has been directed toward the potential effects on human health of the precursor contaminants. People are exposed to acid gases, acid aerosols, and acid fogs, as well as precursor constituents such as sulfur dioxide and nitrogen oxides. These represent the putative concentrations which may or may not be precise.

Ideally we would prefer to know the actual dose received from a given concentration of a pollutant. Unfortunately, this is not possible for most pollutants. Dose due to lead exposure can be assessed from blood lead measurement or blood aminolevulinic acid (ALA). Carbon monoxide dose can be assessed from blood carboxyhemoglobin or exhaled breath analysis. Japanese workers have been exploring the use of hydroxyproline excretion in the urine as an index of exposure to nitrogen dioxide (NO_2) (*1*). This is the only acid precipitation precursor for which there may be a technique to assess dose. This method needs further verification but looks promising. Thus, we must be content at this time to examine the relationships between ambient concentrations and actual human exposures.

Consider first some of the precursors to acid precipitation from a physical-chemical viewpoint. Table 1 lists these. It also contains the molecular weights of the different compounds and the ratio of the molecular weight of the compound to that of sulfur dioxide. This means that if a molecule of SO_2 is converted to these other compounds, the molecular weight will increase but the sulfur content remains the same. Similar relationships can be calculated for NO_2 or CO_2. Again comparisons can be made for equivalent amounts of nitrogen dioxide or carbon dioxide as has been done by the column of ratios. If the fundamental element, such as S, C, or N, is the active material, equivalent exposures can be calculated. Thus it may be more informative to relate to equivalents of sulfur etc., rather than actual concentrations present in the air if one believed that sulfur was the active component.

Much of the concern in the past has been with the amount of sulfate (SO_4^{2-}) present in the ambient air. Current thinking is more inclined to be concerned with the cationic portion rather than the anionic sulfate. The measurements of "sulfate" do not give a complete picture and the reactivity of these compounds which can be very high as with the hydrogen ions from sulfuric acid to relatively innocuous as in sodium sulfate. Animal toxicity studies (*2*) have indicated this gradation of toxicity. This has been confirmed in studies on human subjects (*3,4*). Thus to obtain appropriate data on exposure to these compounds the cationic portion needs to be measured. This requires more sophisticated measurement as a component of future health studies.

As a part of the Six Cities Air Pollution-Health Study, we have developed an operational system (Fig. 1) to monitor the concentrations of sulfuric acid and ammonium sulfate/bisulfate in the ambient air continuously (*5*). This method has depended upon the differential volatilization points of these compounds. We now have more than 2 years of continuous measurements: a year in Watertown, MA; 6 winter months in St. Louis, MO; and 6 warm-season months in Harriman, TN. We note some striking comparisons among these cities. Between March and September, 1984, Harriman experienced 25 separate sulfuric acid "events," defined as 2 hr where H_2SO_4 exceeded 3 $\mu g/m^3$. In a year, only 17 "events" occurred in Watertown, MA. In general, the acid "events" experienced in eastern Tennessee last longer

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Table 1. Molecular weight of selected acids and their salts, and the ratio of their molecular weight to that of the original gas.

	Mol. wt.	Mol. wt./mol. wt. pollutant gas
SO_2	64	1
SO_4^{2-}	96	1.50
H_2SO_4	98	1.53
$(NH_4)HSO_4$	115	1.80
$(NH_4)_2SO_4$	132	2.06
Na_2SO_4	142	2.22
NO_2	46	1
HNO_3	63	1.37
CO_2	44	1
H_2CO_3	62	1.41

and have both higher peak and average H_2SO_4 and total particulate sulfate concentrations. While the instrument can not differentiate $(NH_3)HSO_4$ from $(NH_3)_2SO_4$, it is expected that when H_2SO_4 is detected, the atmosphere is in an "acid" condition and that the remaining sulfate is most likely $(NH_3)HSO_4$. Tables 2–4 summarize the frequency, duration, and intensity of these acid events in Harriman, Watertown, and St. Louis. Preliminary results such as these underscore the need to measure the pH of the aerosol mixture in order to differentiate the health effects potentially associated with ambient exposures to airborne sulfur and other acidic particles.

When considering exposures to air contaminants, the conceptual framework shown in Figure 2 is helpful. Ambient sources emit pollutants that are dispersed and transformed in the atmosphere. Depending on several factors, these contaminants can penetrate into dwellings or buildings. Indoor environments, by virtue of combustion, abrasion, and evaporation, can experience additional contamination from many of the same pollutants as are found outdoors. People, for the most part, are mobile. The exposures to elevated concentrations will be determined by their time-activity patterns. Dose and consequent health effects depend upon many host-dependent factors. It is useful to consider this scheme when discussing the uncertainties of assessing the health effects of acid precursor pollutants.

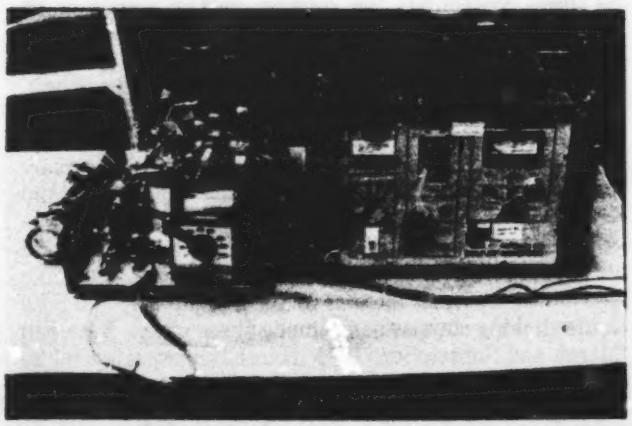

FIGURE 1. Acid analyzer. Heat ramp on left, sulfur analyzer on right.

Table 2. Summary of Harriman, TN, acid aerosol monitoring from March 21, 1984 to September 14, 1984; number of "events" = 25.[a]

	Mean	SD	Maximum
Duration, hr	16.9	17.5	62
Peak 15 min H_2SO_4, $\mu g/m^3$	8.2	4.3	14
"Event" avg H_2SO_4, $\mu g/m^3$	3.5	1.3	7
Peak 15 min TSO_4, $\mu g/m^3$[b]	24.0	8.4	46
"Event" avg TSO_4, $\mu g/m^3$	18.6	6.6	36

[a] H_2SO_4, ($\mu g/m^3$) "event" defined as ≥ 3 $\mu g/m^3$ H_2SO_4 for longer than 2 hr.
[b] TSO_4 = total sulfate.

The acid precursors of sulfur gases and particles are primarily emitted from stationary outdoor sources or are transformed in the atmosphere. However, they are reactive and are measured in lower concentrations indoors. We have found indoor SO_2 concentration to be, on average, only 30% to 50% of the outdoor concentrations. Particle sulfate indoors varies between 50% and 70% of the outdoor concentrations (*6,7*). Of course, there can be considerable seasonal and home-to-home variation in these concentrations. On average, an individual's overall exposure to SO_2 will be less than outdoor ambient because 80–90% of the time we are indoors (*8,9*). Peak exposures, however, are likely to be determined by the time spent outdoors.

For nitrogen dioxides the situation is quite different. Approximately 43 million residences in the United States cook with gas. There are an estimated 13 million kerosene burners in use. Many homes use unvented gas heaters, particularly in less severe climates. As a result, population exposures to NO_2 are strongly influenced by indoor sources (*10,11*). As a result, the determination of actual human exposures to several of the acid precursors is quite complicated. Therefore, it is not surprising that substantial variations in exposures among individuals and subgroup populations exist.

Estimating exposures based on ambient concentrations and categorical variables (gas vs. electric cooking fuel) does not satisfactorily resolve the problem of misclassification. We had originally hoped that merely classifying homes as using gas or electricity for cooking would allow us to characterize the homes. Table 5 shows

Table 3. Summary of Watertown, MA, acid aerosol monitoring from September 16, 1983 to September 14, 1984; number of "events" = 17.[a]

	Mean	SD	Maximum
Duration, hr	14.0	9.1	33
Peak 15 min H_2SO_4, $\mu g/m^3$	8.1	5.5	21
"Event" avg H_2SO_4, $\mu g/m^3$	4.1	1.9	8
Peak 15 min TSO_4, $\mu g/m^3$	22.5	10.5	36
"Event" avg TSO_4, $\mu g/m^3$	16.2	8.6	28

[a] H_2SO_4, ($\mu g/m^3$) "event" defined as ≥ 3 $\mu g/m^3$ H_2SO_4 for longer than 2 hr.

Table 4. Summary of St. Louis, MO, acid aerosol monitoring from October 28, 1983 to March 19, 1984; number of "events" = 12.[a]

	Mean	SD	Maximum
Duration, hr	17.2	18.1	59
Peak 15 min H_2SO_4, $\mu g/m^3$	8.2	10.5	41
"Event" avg H_2SO_4, $\mu g/m^3$	3.7	2.6	11
Peak 15 min TSO_4, $\mu g/m^3$	22.3	13.8	52
"Event" avg TSO_4, $\mu g/m^3$	12.8	4.0	20

[a] H_2SO_4, ($\mu g/m^3$) "event" defined as $\geq 3 \mu g/m^3$ H_2SO_4 for longer than 2 hr.

Table 5. Comparison of estimated week-long integrated exposures to NO_2 versus measurements using personal monitors (Portage, WI, study, approximately 300 adults and children, one week winter, one week summer, N = 594).[a]

Model	R_2	RMSE, $\mu g/m^3$[b]
Ambient NO_2 concentration	0.01	14.9
Model 2 and gas vs. electric stove (yes/no)	0.41	11.8
Model 3 and bedroom concentrations	0.67	8.8

[a] Data of Quackenboss (*12*).
[b] Root mean square error.

that that approach did not predict well, and R^2 values were low. It was not until we actually measured the concentrations in the home and used them to predict exposures that our predicted values became more consistent with actual exposures as measured by personal sampling.

The reason for the discrepancy is shown in Figure 3, where we have plotted the predicted personal NO_2 concentrations in two communities with ambient concentrations of 20 $\mu g/m^3$ and 70 $\mu g/m^3$. It was assumed that 50% of the homes had gas stoves. The model assumed three microenvironments (home, outdoors, work) with no sources in electric cooking homes or at work. Time–activity patterns are derived from several of our studies on personal exposures. Figure 3 demonstrates that considerable overlap in exposures can occur because of indoor gas-cooking, variations in ventilation rates and human activity patterns. Thus if we used merely gas cooking or electric cooking as our dichotomous variable, we would produce much misclassification of our subjects with respect to exposures.

Because of variations in source use, ventilation rates and time–activity patterns, a tenfold spread in annual averaged indoor NO_2 concentrations can occur in homes within the same community (*10*). When considering the additional variation in actual personal exposure due to differences in activity patterns and ambient concentrations, there is the potential for substantial misclassification among subjects. Ozkaynak et al. (*13*), expanding upon the work of Shy et al. (*14*) and others, calculated the effect of misclassification bias on relative risk and chi-squared outcomes from epidemiology. He has demonstrated that effects can go undetected in population studies as a result of even modest misclassification of exposure.

It is further demonstrated that fixed site ambient monitoring may not represent well the actual exposure of human beings for pollutants with indoor sources. It can be questioned how well a single ambient site in a community characterizes the concentrations across the study population. Both NO_2 and SO_2 have spatial gradients on the scale of 100 m to 1000 m. This is more true for SO_2, which is emitted essentially from stationary sources. It is not uncommon to find 2- to 10-fold differences in both short-term and long-term averaged concentrations within an urbanized area. SO_2 and to some degree NO_2 typically have seasonal patterns with higher concentrations in the heating season.

In contrast, sulfate and nitrate particles have spatial patterns that are more uniform over distances of 10 km to 100 km. Their concentrations are higher in the warmer months. These physical characteristics have implications to population exposures. The relationship between measured concentrations and actual human exposure will be more site-dependent for SO_2 and NO_2 and less so for the secondary aerosols. Sulfate, for instance, is more uniform on an urban or regional basis, thus fewer sites can adequately characterize ambient

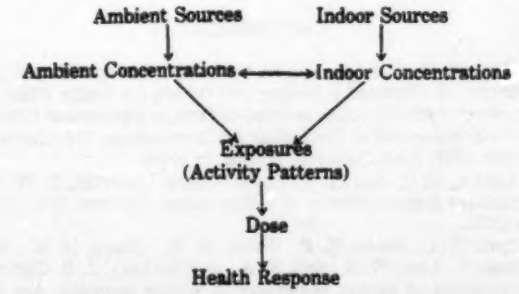

FIGURE 2. Framework for understanding relationship of human exposures and health effects.

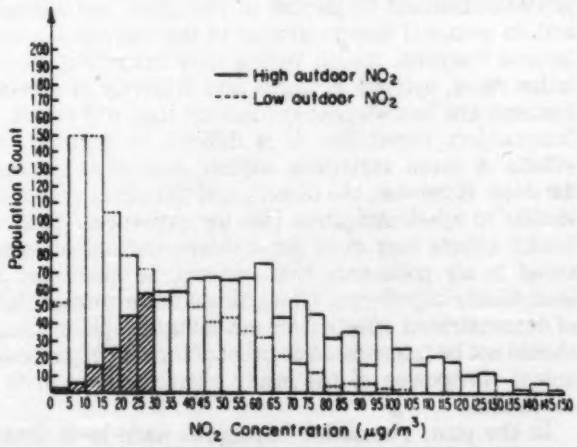

FIGURE 3. Frequency distributions of personal exposure of two groups of adults, one exposed to "high" NO_2 (70 $\mu g/m^3$), the other to "low" NO_2 (20 $\mu g/m^3$).

concentrations. Cross-sectional studies of populations from communities within a geographical region may be different in ambient NO_2, SO_2, and TSP concentrations, but actually may have quite similar sulfate (and ozone) concentrations. Finally, the temporal features associated with these pollutants will modify the potential for population exposure. Both time–activity patterns of people and the residential air exchange rates are a function of season. They vary in such a manner to maximize human exposures to secondary aerosols (sulfates), and to minimize exposures to sulfur dioxide gas.

Another factor influencing exposure and dose of particulate matter is the particle size distribution, since this will determine site of deposition. This is discussed in more detail by Martonen (*15*). In summary, larger mode particles (> 10 μm) tend to be deposited in the nasopharynx and smaller ones (fine mode <2.5 μm) are deposited more deeply in the respiratory tract. In general, smaller particles tend to be deposited more deeply in the lung. Particles in the coarse mode 10–2.5 μm tend to deposit in the bronchial tree. Some particles or droplets such as sulfuric acid can undergo increase in diameter due to the warm humid conditions of the respiratory tract. Thus their deposition will be higher in the respiratory tract than would have been predicted from the ambient air size-distribution. These various factors are also influenced by tidal volume, minute ventilation, and whether or not the individual is mouth breathing. Sulfate aerosols are primarily in the submicron size range. Their size fractionation between fine and coarse particles is 10 to 1. In contrast, nitrate particles are more equally proportioned between fine and coarse modes.

We must not overlook, however, the larger particles (>10 μm) that are likely to be deposited along the nasopharynx. They may have a health effect such as the exposures to wood dust that have resulted in nasal cancers (*16*) or even produced bronchoconstriction and/or rhinitis (*17*).

Hence, dose to target organs will depend both on the physical/chemical properties of the gases and aerosols and on personal factors unique to the individual. Anatomical features, mouth versus nose breathing, ventilation rates, activity patterns and integrity of defense systems are host-dependent factors that will result in intersubject variability. It is difficult to quantify the effects of these variations without biological markers for dose. However, the direction of the effect should be similar to misclassification bias for exposures. That is, health effects may exist for ambient and indoor exposures to air pollutants that can not be quantified as statistically significant. Given these uncertainties, lack of demonstrated effects from air pollution epidemiology should not be interpreted as proof of the null hypothesis, unless the power of the study readily allows such a conclusion.

In the past, population exposures have been determined by single monitoring stations that are used to reflect exposure over a radius of 1 to 5 km. For some sites this may be true, but it should be verified by actual measurements. Frequently the sites chosen have been selected primarily for regulatory purposes or because they represent "hot spots." For epidemiologic purposes, it would be more appropriate to have monitoring sites where the populations are. These may be in addition to the regulatory sites. If "hot spots" are used to reflect exposures there will be misclassification of exposure and if no health effects are seen this could be attributed to a higher concentration than it should be. These points indicate a need for more personal monitoring to attain better estimates of exposure. It should be pointed out that personal sampling has its problems. Some of these have been commented on by Fugas (*18*). They can be summarized as follows.

Personal monitors are not available for many of the pollutants. Presently they are available for CO, SO_2, NO_2, formaldehyde and a few other gaseous pollutants.

Analysis of the samples can be time-consuming and expensive.

The sample of the population may not be representative of the general population, as many industries will not allow such devices on their premises. The samplers may prove to be cumbersome or annoying so other persons will not wear them.

The wearing of a sampler may modify the behavior of the person so that the results are not truly representative of that person. This might be controlled for by having the individuals keep activity diaries both when wearing the samplers and when not wearing them.

Thus, to estimate the exposure and dose to a pollutant, we need to assure ourselves of the following: (1) that the outdoor monitors are indeed representative of the outdoor exposure; (2) that the outdoor concentrations have been corrected to reflect exposure indoors (probably best done by actual measurements indoors and an activity diary, or by personal sampling); (3) that particle size distributions are described as well as the chemistry of the particles.

From this comes the need to develop lightweight, accurate and reliable personal monitors to assess more precisely the individual and then hopefully the population exposure.

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